

Literature Review: The Effect of Childhood Trauma on Cortisol Levels

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Introduction

Childhood trauma is a very important and impactful issue. Nearly 46% of children 17 and under experience some sort of trauma (“Helping Children”, n.d.). One of the ways this can impact children is through changing the ways their bodies regulate stress. In the human body, cortisol, a steroid hormone, is released from our adrenal glands when we are feeling stressed. It helps regulate our physiologic response system, along with being important to maintain proper metabolism. When this response system is overused, children can experience serious physical and mental complications, such as cognitive difficulties and anxiety disorders (Nader and Weems, 2010). The research for how childhood trauma impacts cortisol production and stress regulation is a bit complicated, as many studies have gathered mixed results. Generally, there are two common trends: childhood trauma either leads to increased or blunted cortisol production. Sometimes, there is even no correlation between trauma and cortisol levels. This review will dig deeper into the nuances of how trauma influences regular cortisol functioning, and will work to synthesize the currently available research.

Why does cortisol matter?

When we experience a stressful situation, our hypothalamic-pituitary-adrenal (HPA) axis releases the corticotropin-releasing hormone (CRH) which signals the pituitary gland to release the adrenocorticotropic hormone (ACTH). The ACTH eventually reaches the top of the kidneys where the adrenal glands are located (Fogelman and Canli, 2018). The adrenal glands will release cortisol, which increases the brain’s availability for blood glucose (Thau et al., 2021). The blood glucose allows for the brain to generate ATP, providing us with more energy and increased thinking ability. This is what the human body normally does when we encounter a stressor.

Generally, there are two patterns that emerge in regards to the stress regulation system when the body experiences chronic stress. To start off, increased stress response system activation can cause cortisol levels to increase. Overuse of the stress response system may lead to changes which cause dysregulation, particularly in children, who’s regulatory systems are still developing. One way this occurs is that cortisol increases as ACTH increases (Herman et al., 2016). This would lead to more cortisol being produced due to the lack of inhibition for the ACTH, and works as a positive feedback loop due to less regulation. If this were to happen chronically during a child’s “critical”, or early development periods, the stress response system’s development may show signs of dysregulation. Dysregulation of this system has been associated with chronic illnesses, such as depression, psychological “burn out”, and heart disease (Fogelman and Canli, 2018).

Alternatively, chronic stress can also lead to suppression of the stress response system. The HPA axis can be regulated by glucocorticoids which inhibit CRH production (Tsigos and Chrousos, 2002), working as a negative feedback system to prevent too much cortisol from being produced. This is due to the fact that CRH is what signals the pituitary gland to release ACTH, which leads to cortisol production after ACTH reaches the adrenal glands, as explained earlier. The inhibition of this would lead to lowered, or blunted cortisol. If this were to occur chronically, especially when a child’s stress response system is still developing, it would also lead to signs of dysregulation. To add on, blunted cortisol levels can lead to issues with proper brain functioning (Diseth, 2005). For example, the overuse of the HPA axis for cortisol purposes

in children can cause them to experience cognitive decline as they reach adulthood (Gaysina et al., 2014).

In the long term, under or over activation of the stress response system has been associated with negative outcomes.

How is childhood trauma related to cortisol?

There are different ways in which cortisol can react and adapt in response to chronic childhood trauma. Many studies indicate that the presence of childhood trauma in an individual's past leads them to have increased cortisol levels. As explained earlier, constant stress during childhood may lead to abnormal stress response system development. According to a study by Jaffee et al. (2014), children who experienced traumatic events early in life had increased cortisol reactivity. Another study found that childhood trauma specifically caused by injuries, led to higher cortisol reactivity if the injuries were severe (Okur et al., 2007). Larger environmental contexts associated with stress can also impact cortisol levels. For example, socioeconomic hardships have been associated with increased cortisol levels.

However, there are many studies that suggest that increased trauma and stress is associated with decreased cortisol levels. Often, childhood trauma may lead to PTSD later in life. PTSD has been associated with a blunted ACTH response, which leads to less cortisol production in response to stress (King et al., 2001). One cortisol-testing study consisted of individuals with and without childhood trauma. The cortisol levels were lower than usual in the individuals who had a history of childhood trauma, compared to the ones who did not (Suzuki et al., 2014). Another study which was specifically about sexual abuse histories during childhood displayed that individuals who were sexually abused within the last couple of months had lower cortisol than usual. The study also pointed to a conclusion that the children had impaired HPA axis functioning after early childhood trauma (King et al., 2001). Lastly, an interesting study that was based on incarcerated adults discovered that the adults with childhood trauma who had psychopathic tendencies showed lower cortisol levels than typical (Cima et al., 2008). While this may not be due to direct causation, it is an interesting connection that points towards how low cortisol may be related to worsened mental health, and perhaps childhood trauma may have influenced the development of such mental health challenges.

While the two most common patterns present in the studies are that low or high cortisol is the result of childhood trauma exposure, other studies have shown that childhood trauma may not significantly affect cortisol levels. The lack of relation between trauma and cortisol in these studies could indicate the presence of regular functioning of feedback systems where ACTH inhibition and CRH production are able to occur at the right times. According to Fogelman and Canli (2018), the presence of early life stress and trauma did not significantly impact the cortisol responses. In a study in 2009 by Flinn, he discovered that traumatic events during critical periods of a child's youth did not appear to cause permanently elevated cortisol levels. This displays that childhood trauma does not always cause elevated or blunted cortisol levels.

Together, the findings are mixed. Many studies suggest that there are increased cortisol levels after childhood trauma exposure, others suggest decreases, and some point to no effect at all. While a lot of this is due to the different adaptations from the stress response system, some may be caused by experimental factors. For instance there are multiple methods to measure cortisol, such as saliva. However, salivary cortisol is less reliable and isn't the most informative measure to understand long term HPA activity. The cortisol measurements of many studies through salivary cortisol may give different results rather than something like hair

cortisol, which gives a superior overall measure of HPA activity since it avoids methodological problems in comparison to salivary and other types of cortisol measurements. Contamination and timing of samples isn't as big of an issue when measuring hair cortisol. Cortisol measured from hair closest to the scalp also has a long-term benefit as it gives cortisol measurement estimations for up to six months. (D'Anna-Hernandez et al., 2011).

Does it vary based on trauma?

There are many different types of traumas that vary widely in their extremity and persistencies. It is possible that the different forms of trauma may be related to cortisol production in different ways. Certain types of traumas may be more or less detrimental on HPA axis functioning. For example, participants who experienced physical abuse specifically had noticeably blunted cortisol levels. According to Carpenter et al. (2011), women who self-reported childhood physical abuse had decreased cortisol levels. However, in another study by Flory et al. (2009), while individuals with histories of physical abuse also displayed decreased levels of cortisol, those with physical neglect histories had increased cortisol levels instead. More variation is also prominent in sexual and emotional-related traumas. In sexual abuse, while there were some studies which displayed decreased cortisol levels, most of them pointed towards an increase. For example, in an study by Şimşek et al. (2015), individuals who went through sexual abuse had cortisol levels that were higher than normal, and the same type of results for sexual abuse were present in the study by Carpenter et al. (2011) that was mentioned previously. According to Yehuda et al. (2001), individuals who went through sexual abuse, but did not have PTSD, had significantly higher cortisol levels. As shown, those with histories of sexual abuse generally have higher cortisol levels. As for emotional-related traumas, according to the same study by Yehuda et al. (2001), emotionally abused subjects had lower cortisol levels in comparison to those who were sexually abused. Similar results were present in a different study by Carpenter et al. (2009), displaying that emotionally abused subjects had diminished cortisol responses. As for emotionally neglected individuals, they had similar, lowered cortisol responses (Carpenter et al., 2011), similar to the abused. There is a lot of variation between the types of traumas and what kind of results are shown.

Similar patterns may be present but there also may be outliers. A lot of this may be due to experimental related factors or the subjects' current mental states, possibly altering results. For example, if two individuals in a study had similar histories of childhood trauma but one had PTSD, they may show completely different cortisol levels. The current mental state of the individual is something to take into account in these types of studies since other disorders or life issues may also be impacting their stress response system during the measuring.

Conclusions

Future Directions of the Field

Future research should continue to explore the relation between childhood trauma and cortisol levels. A way to improve the research is to determine the mental state of the subjects, or have separate trials for those with depression, PTSD, etc. This would help further understand the relation between these types of disorders that can be caused by childhood trauma with cortisol levels. Also, the differences between the measurements of hair cortisol and salivary cortisol should be compared to understand the patterns of cortisol levels between the two.

Limitations



Many studies exploring this relation have limitations, including the amount of individuals, the current mental state of the individuals, etc. As explained previously, the relation between different disorders caused by childhood trauma may have different effects on cortisol levels if they are present in the subject or not. This isn't taken into account as often, but may still play an important role. Also, a lot of studies are performed with a limited number of subjects, leading to possibly different, uncorrelated results rather than a study with a larger number of subjects. For example, a particularly good study by Jaffee et al. (2015) included 400 children and also took into account the HPA axis' functioning in relation to the cortisol levels in the discussion. This would give a better and more accurate understanding of the relation of childhood trauma and cortisol levels. Generally, there are many things that could impact the results making it so we don't have one clear answer to how childhood trauma really affects cortisol levels, so we have variation instead.

Takeaways

Due to childhood trauma, cortisol may change or adapt our stress regulation systems and also may impact the functioning of our HPA axis. Often, we see that there is either higher or lower cortisol in the long term based on childhood trauma's presence in an individual. There is a lot of variation between all these different results, and certain studies even suggest that there is no correlation between childhood trauma and cortisol levels. The variation in results may be due to experimental factors and the way that the stress response system differently adapts. There are also certain patterns and differences displayed through the many types of trauma, such as physical and emotional abuse showing lower cortisol levels, whereas sexual abuse generally displays higher levels. The relation between childhood trauma and cortisol levels later in life should continue to be researched to give us a greater understanding of this important connection.

References

1. Carpenter, L. L., Shattuck, T. T., Tyrka, A. R., Geraciotti, T. D., & Price, L. H. (2011). Effect of childhood physical abuse on cortisol stress response. *Psychopharmacology*, *214*(1), 367-375. <https://doi.org/10.1007/s00213-010-2007-4>
2. Cima, M., Smeets, T., & Jelicic, M. (2008). Self-reported trauma, cortisol levels, and aggression in psychopathic and non-psychopathic prison inmates. *Biological psychology*, *78*(1), 75-86. <https://doi.org/10.1016/j.biopsycho.2007.12.011>
3. D'Anna-Hernandez, K. L., Ross, R. G., Natvig, C. L., & Laudenslager, M. L. (2011). Hair cortisol levels as a retrospective marker of hypothalamic–pituitary axis activity throughout pregnancy: comparison to salivary cortisol. *Physiology & behavior*, *104*(2), 348-353. <https://doi.org/10.1016/j.physbeh.2011.02.041>
4. Diseth, T. H. (2005). Dissociation in children and adolescents as reaction to trauma—an overview of conceptual issues and neurobiological factors. *Nordic journal of psychiatry*, *59*(2), 79-91. <https://doi.org/10.1080/08039480510022963>
5. Fogelman, N., & Canli, T. (2018). Early life stress and cortisol: A meta-analysis. *Hormones and Behavior*, *98*, 63-76. <https://doi.org/10.1016/j.yhbeh.2017.12.014>
6. Gaysina, D., Gardner, M. P., Richards, M., & Ben-Shlomo, Y. (2014). Cortisol and cognitive function in midlife: the role of childhood cognition and educational attainment. *Psychoneuroendocrinology*, *47*(100), 189–198. <https://doi.org/10.1016/j.psyneuen.2014.05.018>
7. Helping Children and Youth Who Have Traumatic Experiences. (n.d.) *Substance Abuse and Mental Health Services Administration*. https://www.samhsa.gov/sites/default/files/brief_report_natl_childrens_mh_awareness_da_y.pdf
8. Herman, J. P., McKlveen, J. M., Ghosal, S., Kopp, B., Wulsin, A., Makinson, R., Scheimann, J., & Myers, B. (2016). Regulation of the Hypothalamic-Pituitary-Adrenocortical Stress Response. *Comprehensive Physiology*, *6*(2), 603–621. <https://doi.org/10.1002/cphy.c150015>
9. King, J. A., Mandansky, D., King, S., Fletcher, K. E., & Brewer, J. (2001). Early sexual abuse and low cortisol. *Psychiatry and clinical neurosciences*, *55*(1), 71-74.
10. Nader, K., & Weems, C. F. (2011). Understanding and assessing cortisol levels in children and adolescents. *Journal of Child & Adolescent Trauma*, *4*(4), 318-338. <https://doi.org/10.1080/19361521.2011.624059>
11. Okur, H., Küçükaydn, M., Özokutan, B. H., Muhtaroglu, S., Kazez, A., & Turan, C. (2007). Relationship Between Release of β -Endorphin, Cortisol, and Trauma Severity in Children With Blunt Torso and Extremity Trauma. *Journal of Trauma and Acute Care Surgery*, *62*(2), 320-324. <https://doi.org/10.1097/01.ta.0000222714.91463.a3>
12. Suzuki, A., Poon, L., Papadopoulos, A. S., Kumari, V., & Cleare, A. J. (2014). Long term effects of childhood trauma on cortisol stress reactivity in adulthood and relationship to the occurrence of depression. *Psychoneuroendocrinology*, *50*, 289-299. <https://doi.org/10.1016/j.psyneuen.2014.09.007>
13. Thau, L., Gandhi, J., & Sharma, S. (2021). Physiology, cortisol. In *StatPearls [Internet]*. StatPearls Publishing.



14. Tsigos, C., & Chrousos, G. P. (2002). Hypothalamic–pituitary–adrenal axis, neuroendocrine factors and stress. *Journal of psychosomatic research*, 53(4), 865-871. [https://doi.org/10.1016/s0022-3999\(02\)00429-4](https://doi.org/10.1016/s0022-3999(02)00429-4)